to be lower than in status 2 candidates. The reasons for this difference could not be explained by our data and further investigations are required. In order to maintain status 1 candidature for pts who otherwise would need urgent HTx, optimal management of pretransplant therapy could include elective assist device implantation.

1097-57 Four Year Experience With the Functional and Morphological Adaptation of Undersized Donor Hearts Following Cardiac Transplantation

P.J. Mather, V. Jeevanandam, A.A. Bove. *Temple University School of Medicine, Philadelphia, Pennsylvania, USA*

We compared our experience with the transplantation of 71 undersized donor hearts over a four year period to a cohort group of 71 normal sized donor hearts. The donor to recipient weight ratios were 0.65 ± 0.08 for the undersized hearts, and 0.96 ± 0.08 for normal sized hearts. Left ventricular mass (LVM) was obtained by echocardiography. Hemodynamics and VO_2 max were obtained at yearly intervals.

	10 Weeks		One Year (N = 71)		Four Years	
	Undersized	Normal	Undersized	Normal	Undersized	Normal
VO_2	13.0 ± 2.4	14.6 ± 3.5	0.8 ± 3.0	20.1 ± 3	23.3 ± 5.4	24.0 ± 4
PVR	3.7 ± 0.8	2.4 ± 0.8	2.8 ± 0.6	2.6 ± 0.6	2.8 ± 0.7	2.7 ± 1.0
Survival	100%	100%	91.5%	80.0%	70%	77%
LVM	195 ± 24	160 ± 30	190 ± 32	155 ± 40	184 ± 35	166 ± 30

VO_2 = oxygen consumption exercise test (ml/kg/min). PVR = pulmonary vascular resistance (Wood units); LVM = in grams. N = 47 for undersized at four years, and N = 52 for normals at four years

The increase in LVM in the undersized hearts, and then their subsequent plateau over time suggest that the left ventricle adapts to the larger recipient circulation. The functional capacity of the two groups, hemodynamics, and survival rates are not significantly different. Therefore, despite denervation and a mismatched load, transplanted hearts adapt appropriately to their new hemodynamic milieu, thus suggesting the cardiac donor pool can be expanded to include undersized hearts.

1097-58 Can Anti-HLA Antibodies Post Cardiac Transplantation Predict Clinical Outcome?

G. Bhat, T.J. Schroeder, K. Balakrishnan. *University of Cincinnati, Cincinnati, OH, USA*

Background: The panel reactive antibody (PRA) test is used for HLA antibody screening in transplantation. The purpose of this study was to determine anti-HLA class I IgG antibodies by ELISA-based assay (PRA-STAT) post-cardiac transplant (PCT) and correlate with clinical outcome.

Methods: Pre and PCT sera from 107 patients (pts) undergoing primary cardiac transplantation were retrospectively analyzed. All pts received an OKT3 immunosuppressive protocol followed by triple therapy; graft survival was 80.4% with a mean follow up of 45 months (range 16–79). Clinical outcomes were analyzed as episodes of ≥3A grade acute rejection (AR) within 31, 60, and 90 days, time (days) to first episode of AR, graft loss within 60 days and overall graft loss.

Results: Pts with PCT PRA-STAT at day 10 <10% vs ≥10% had 39% compared to 14.6% overall graft loss (p = 0.041), and 16.6% compared to 2.6% graft loss within 60 days (p = 0.048). 41 pts experiencing AR within 31 days had a significantly higher PRA-STAT (10.2 ± 2.4% vs. 3.3 ± 2.2%, p = 0.038) compared to 48 pts with no AR. Pts with PRA-STAT at day 10 PCT <15% vs. ≥15% had first AR at 22.8 ± 12.4 days compared to 48.9 ± 4.5 days (p = 0.052). Similar PRA-STAT results were observed with day 20 and 30 samples. Increasing PRA during the first month was associated with a significant increase in AR.

Conclusions: PRA-STAT results PCT correlated with early and late graft loss and incidence of acute rejection.

1098 Ventricular Arrhythmias: Remodeling

Tuesday, March 31, 1998, 9:00 a.m.–11:00 a.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 10:00 a.m.–11:00 a.m.

1098-173 Ectopic Beats set the Stage for Torsade de Pointes Arrhythmias by an Important Contribution to Interventricular Dispersion

B. Göreneck, C. Verduyn, M.A. Vos, H.J.J. Wellens. *Cardiology, Maastricht, The Netherlands*

Usually, a short long short sequence (SLS) caused by ectopic beats (EB) precedes the onset of Torsade de Pointes arrhythmias (TdP). Because the coupling interval of EBs often falls within the QT or action potential duration (APD), their role in the dynamics of (dispersion of) repolarization is difficult to assess. In 17 anesthetized dogs with complete chronic AV block, we measured APD_{100} and APD_{50} of the left and right ventricle (LV, RV) using endocardial monophasic action potential catheters. Administration of class III agents almokalant (n = 9) or ibutilide (n = 8) resulted in spontaneous TdP. Dispersion (ΔAPD) was defined as LV APD-RV APD and was determined for the last normal beat prior to TdP (beat I), the beat followed by EB but not resulting in TdP (II) and the beat directly preceding TdP (III).

Results: The class III drugs increased APD, the % increase of the APD_{100} and APD_{50} was comparable in both ventricles (r = 0.72, p < 0.001). In 7 dogs the TdP started sudden i.e. not preceded by SLS. These dogs had a significantly longer ΔAPD at beat I, than the non sudden group (table). In the non sudden group, the sequence of EBs increased the LV APD but not the RV APD leading to a similar ΔAPD as the sudden onset (table).

	Sudden onset (n = 7)	Non sudden onset (n = 10)		
	beat I	beat I	beat II	beat III
LV APD_{50}	510 ± 33	395 ± 32	419 ± 33	450 ± 32*
RV APD_{50}	380 ± 26	330 ± 36	332 ± 35	320 ± 43
ΔAPD_{50}	130 ± 12*	66 ± 13	86 ± 25*	130 ± 32*

*p < 0.05 vs beat I non sudden onset

Conclusions: When the magnitude of ΔAPD is not large enough for one EB to trigger TdP, the sequence of EBs can further increase ΔAPD and thereby set the stage for TdP.

1098-174 Left Ventricular Hypertrophy Increases Vulnerability to Bradycardia and D-Sotalol Induced Early Afterdepolarizations

S.J. Rials, Y. Wu, T. Liu, X. Xu, D.D. Bharucha, R.A. Marinchak, P.R. Kowey. *Lankenau Hospital and Medical Research Center, Wynnewood, PA, USA*

Left ventricular hypertrophy (LVH) is known to prolong action potential duration (APD) and increase the risk of ventricular arrhythmia, possibly by generating early afterdepolarizations (EAD). If true, then LVH preparations should be more vulnerable to class III induced EAD. We examined whether APD prolongation by LVH leads to EAD using transmembrane action potentials recorded in arterially perfused wedges of ventricles from rabbits with LVH. Recordings were made at multiple pacing cycle lengths (PCL), both pre and post perfusion with 1 μM d-sotalol. LVH preparations showed greater prolongation of APD at longer PCL in a reverse use dependent manner, both before and after d-sotalol (p < 0.05 vs control). 3 of 8 LVH preparations showed EAD pre sotalol and 7 of 8 LVH preparations showed EAD post sotalol. None of the control preparations showed EAD either before or after sotalol (p < 0.05 vs LVH). EAD in the LVH preparations were observed to trigger action potentials in surrounding tissue and to cause premature beats on transmural ECG. We conclude that LVH increases vulnerability to bradycardia and to d-sotalol induced EAD. These EAD can reach threshold and propagate to surrounding tissue. These data demonstrate that drugs that increase action potential duration may promote ventricular proarrhythmia in LVH by increasing vulnerability to EAD.

1098-175 Effects of Quinaprilat Electrophysiologic Changes During Acute Myocardial Ischemia and Following Reperfusion in Canines

H. Fujinaga, T. Wakatsuki, K. Sakabe, J. Ikata, T. Toyoshima, A. Nishikado, T. Oki, S. Ito. *Tokushima University, Tokushima, Japan*

Purpose: We examined the effects of angiotensin converting enzyme inhibitor as quinaprilat on the electrophysiologic changes during acute myocardial ischemia and following reperfusion.

Methods: The left anterior descending (LAD) coronary artery was ligated for ten minutes and then reopened for ten minutes in 33 canines. They were divided into two groups (the quinaprilat group; 14, the control group; 19). The quinaprilat group received quinaprilat by an infusion of 3 μ g/kg/min from 10 min before the ligation to the end of examination. The control group received normal saline. We compared changes in the ventricular effective refractory period (ERP) of LAD region during myocardial ischemia and following reperfusion, and incidence of ventricular fibrillation (Vf) between the two groups. ERP values during procedure were divided by those before coronary ligation, and expressed as percentages.

Results: ERP of both groups were as follows:

Occlusion time (min)	2	4	6	8	10
Quinaprilat group	101 ± 8	103 ± 8	102 ± 9	105 ± 11	106 ± 14
Control group	97 ± 7	96 ± 9*	92 ± 9**	88 ± 9***	91 ± 11**

TUESDAY POSTER